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International patterns and trends in testis cancer incidence

Mark P. Purdue*, Susan S. Devesa, Alice J. Sigurdson and Katherine A. McGlynn

Division of Cancer Epidemiology and Genetics, National Cancer Institute, Bethesda, MD, USA

Although the incidence of testis cancer has risen markedly in many Western populations over the past half-century, it is not clear whether rates in other populations also have increased. To clarify this issue, we examined testis cancer incidence rates over the 25-year time period of 1973–1997 for selected populations around the world. Age-standardized incidence rates for 21 registries in the Americas, Asia, Europe and Oceania over successive 5-year time periods were obtained from volumes 4–8 of *Cancer Incidence in Five Continents*. Testis cancer rates rose between 1973 and 1997 in most populations worldwide, although the increases were strongest and most consistent among populations of European ancestry. Rates appear to be leveling off in some populations. The increases in testis cancer remain unexplained, although changes in the prevalence of important risk factors for this disease may be responsible.
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Testis cancer is a relatively rare neoplasm worldwide, accounting for approximately 1% of all male cancers diagnosed.[1] However, in more developed countries, this cancer is the most commonly diagnosed malignancy among men aged 15–44. The international incidence of testis cancer varies considerably. Rates are highest in Scandinavian populations, and rates also are elevated among other populations in Europe or of European ancestry. By contrast, Asian and African populations, including U.S. blacks, experience very low rates.[2]

Among many Western populations, testicular cancer incidence increased markedly over much of the past 20th century,[3,4] although recent data from some cancer registries suggest that rates may be plateauing.[5,6] A divergence in trends for different histologic subtypes of testis cancer has also been reported. The majority of testis cancers are germ-cell tumors, which are grouped histologically into seminomas and nonseminomas.[3] In Canadian and U.S. white populations, the incidence of nonseminoma has been increasing less rapidly than that of seminoma.[7,8] No adequate explanations for these trends have been identified, as the etiologies of these malignancies remain largely unknown.

As investigations into secular trends of testis cancer have generally focused on populations of European ancestry, it is not clear whether rates in low-incidence populations also have increased over time. A study on international cancer trends between 1973–1987 by the International Agency for Research on Cancer (IARC) clearly demonstrated rising testis cancer rates in Japan and Puerto Rico but not in other low-incidence registries.[4] Since that time, IARC has published rates for additional time periods, enabling the analysis of secular trends over a longer time span.[2,9] To better understand how testis cancer incidence has changed over time across different populations, we examined incidence data over the 25-year period 1972–1997 from 21 populations in the Americas, Asia, Europe and Oceania.

Material and methods

Incidence data

To examine the secular trends in the incidence of testis cancer (ICD-8, ICD-9 186),[10,11] age-standardized (World Population)[12,13] incidence rates in 21 populations were obtained from volumes 4–8 of *Cancer Incidence in Five Continents* (CI5).[2,9,14,15,16] The CI5 volumes include incidence data reported by selected population-based cancer registries covering areas within Asia, Oceania, Africa, Europe and the Americas. Volumes 4–8 generally provided data for the 5-year time periods 1973–1977, 1978–1982, 1983–1987, 1988–1992 and 1993–1997, respectively. Rates for volumes 5–8 were based upon the ICD-9 classification of testis cancer, and volume 4 used the ICD-8 classification; there were no changes in the coding of testis cancer between the eighth and ninth ICD revisions. Incidence rates for different histologic subtypes of testis cancer (seminoma, nonseminoma, other/unspecified) were abstracted from volume 8.

Populations were chosen for inclusion in our analysis on the basis of the following criteria: *(i)* the availability of rates in CI5 for time periods at least as far back as 1978–1982; *(ii)* an absence of changes in population coverage or of warnings regarding data quality reported in CI5 volumes 4–8; and *(iii)* a sufficiently large number of registered cases in CI5 volume 8 to enable analyses of recent rates by histologic subtype (trends by histologic subtype are not included in our study). Only one registry from each country was selected; if more than one registry met the basic criteria, the registry with the largest population was included in the analysis. A total of 21 populations were selected: 5 from the Americas, 4 from Asia, 4 from the Nordic countries, 5 from elsewhere in Europe (hereafter referred to as ''Europe'') and 3 from Oceania. No African or South Asian populations met the inclusion criteria. However, 5 African registries included in CI5 volume 8 (Algeria, Algiers; France, La Reunion; Mali, Bamako; Uganda, Kyadondo Country; Zimbabwe, Harrare) were included in the comparison of international rates for the 1993–1997 time period, as was the registry of Mumbai, India.

Incidence rates for the U.S. Surveillance, Epidemiology and End Results (SEER) white and black populations were not included in CI5 volumes 4 and 5. The SEERStat statistical package was used to calculate rates for these populations for the relevant time periods.[17]

Data analysis

Trends in age-standardized (World Standard) incidence rates were examined for the time periods 1973–1977, 1978–1982, 1983–1987, 1988–1992 and 1993–1997. Testis cancer rates from New Zealand were reported in CI5 separately for Maori and non-Maori populations for all time periods except 1993–1997, for which only overall rates were reported. For these populations, trends were examined up to 1988–1992.

The percentage change in testis cancer rates between 1973–1977 and 1993–1997 was calculated for each population to show the relative change in incidence between these 2 time periods. Graphs plotting the trends in age-standardized incidence across the 5 time periods for each population were prepared using a semi-log scale to facilitate the comparison of temporal trends as well as magnitude.[18] Graphs describing trends in age-standardized

***Correspondence to:** Division of Cancer Epidemiology and Biostatistics, National Cancer Institute, EPS 8121, 6120 Executive Blvd., Bethesda MD 20892-7240, USA. Fax: +301-402-1819.
E-mail: purduem@mail.nih.gov
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TABLE I – INTERNATIONAL VARIATION IN TESTIS CANCER INCIDENCE RATES, 1973–1977 TO 1993–1997

Population	1973–1977		1993–1997		Percent change
	Cases	Rate[1]	Cases	Rate[1]	
Europe, Nordic countries					
Denmark	736	5.8	1,481	9.9	70.7
Norway	441	4.4	982	8.2	86.4
Sweden	634	3.0	1,148	5.0	66.7
Finland	241	1.7	363	2.7	58.8
Europe, other					
France, Bas-Rhin	50	3.5	228	7.9	125.7
Switzerland, Geneva	48	5.1	74	6.5	27.5
UK, England, South Thames	545	3.4	1,031	5.4	58.8
Italy, Varese	19	2.3	95	3.9	69.6
Spain, Zaragoza	33	1.7	40	2.2	29.4
Oceania					
New Zealand, Maori	24	4.3	61[2]	7.1[2]	65.1
New Zealand, non-Maori	306	4.5	479[2]	5.6[2]	24.4
Australia, New South Wales	386	3.0	887	5.1	70.0
Americas					
USA, SEER: white	1,625	3.8	3,202	5.6	47.4
Canada, Ontario	303	2.7	1,411	4.4	63.0
Colombia, Cali	26	1.5	79	1.9	26.7
USA, Puerto Rico	34	0.5	55	1.6	220.0
USA, SEER: black	34	0.8	81	1.0	23.5
Asia					
Israel: Jews	134	1.9	377	3.3	73.7
China, Hong Kong	114	1.4	302	1.7	21.4
Japan, Osaka Prefecture	184	0.8	290	1.2	50.0
Singapore: Chinese	33	0.8	86	1.0	25.0
India, Mumbai[3]			227	0.8	
Africa					
Algeria, Algiers[3]			38	0.7	
France, La Réunion[3]			5	0.7	
Mali, Bamako[3]			4	0.7	
Uganda, Kyadondo County[3]			6	0.5	
Zimbabwe, Harrare[3]			12	0.4	

[1]Rate is age-standardized to the world population, per 100,000 person-years.–[2] Counts, rate for 1988–1992 period.–[3] These registries were excluded from the trends analysis due to an absence of data for early time periods, changes in population coverage and/or data quality issues.

incidence for specific age groups (<30 years, 30–49 years, 50+ years) were also created; these rates were calculated using the incidence rates and World Population weights for 5-year age groups published in the CI5 volumes.

Results

The 1993–1997 age-adjusted testis cancer incidence rates varied 10-fold across populations (Table I and Fig. 1), with the highest rate in Denmark (9.9 cases per 100,000) and the lowest in Harrare, Zimbabwe (0.4 cases per 100,000). The highest testis cancer rates were concentrated among Nordic countries. High testis cancer rates occurred among populations of Europe, Oceania and North America, whereas low rates were evident in Latin American, Asian and African populations, including U.S. blacks. Considerable variation in testis cancer incidence among populations was present for every continent but Africa and Oceania. This variation was particularly wide among registries of the Americas; rates were relatively high among the U.S. white and Ontario populations (5.6 and 4.4 per 100,000, respectively) and considerably lower for the populations of Cali (1.9), Puerto Rico (1.6) and for U.S. blacks (1.0).

For most populations, the incidence of seminoma was slightly greater than that of nonseminoma. The proportion of cancers classified as seminoma did not differ systematically between high-incidence and low-incidence populations. The majority of testis cancers registered among the African registries of Mali, Uganda and Zimbabwe were of nongerm-cell/unspecified histology; however, the small numbers of diagnosed cases in these registries (4, 6, 12, respectively) preclude any meaningful inferences.

Testis cancer rates increased from 1973–1977 to 1993–1997 (Table I, Fig. 2); on average, populations experienced about a 60% increase. The largest increases appeared in Puerto Rico and Bas-Rhin (220% and 126%, respectively), whereas the rates for Hong Kong, U.S. blacks and New Zealand non-Maori rose only by approximately 25%. The rates for all Nordic countries increased by a relatively large amount (59–86%). Excluding Puerto Rico, the rates of low-incidence populations generally increased more modestly than those of high-incidence populations.

The testis cancer incidence rates of each population during the 25-year period 1973–1997 are plotted in Figure 2. A fairly consistent increasing trend in rates across time periods was apparent for most populations (Fig. 2). For some of the high-incidence populations (U.S. whites, Ontario and Varese), rates appear to plateau during the most recent time periods. Some trends were nonlinear (*e.g.,* Zaragoza, New Zealand Maori, U.S. blacks, Singapore Chinese), perhaps due to low numbers of cases. Trends also were nonlinear for Hong Kong and Osaka; however, these rates are based on relatively large numbers of cases and are unlikely to be due to chance fluctuations. In Hong Kong, testis cancer rates dropped until the 1982–1987 period and increased thereafter. Osaka rates were stable from 1978–1982 to 1988–1992, with rates lower both between 1973–1977 and 1993–1997.

Trends in age-standardized incidence among males aged 0–29 years, 30–49 years and 50+ years were plotted for 12 populations of sufficient size to provide stable rates (Fig. 3). Testis cancer rates

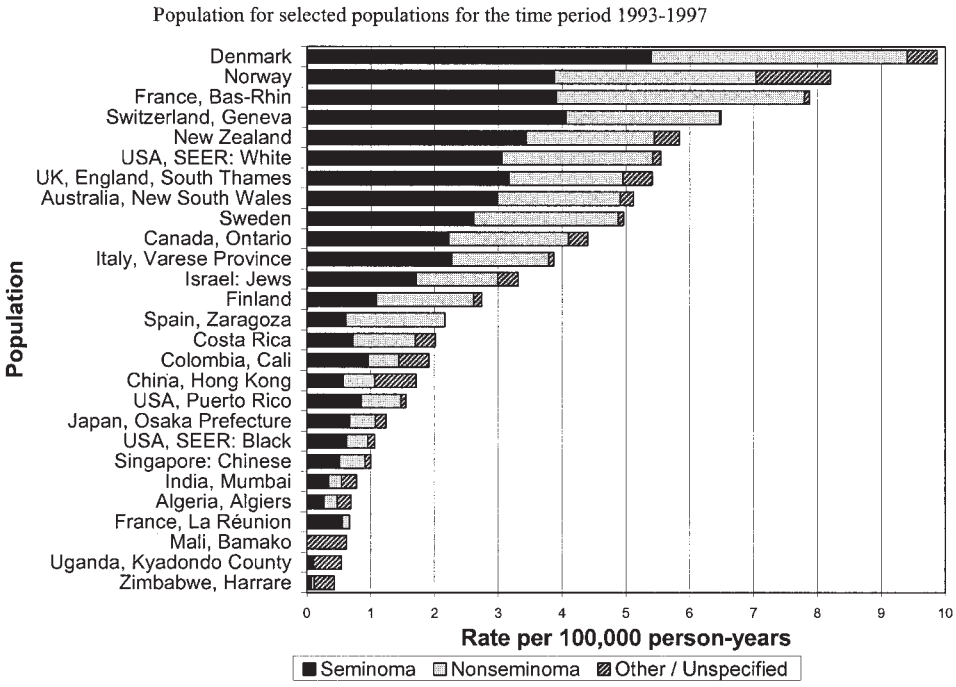

FIGURE 1 – Incidence rates of testis cancer (per 100,000 person-years) age-standardized to the world population for selected populations for the time period 1993–1997.

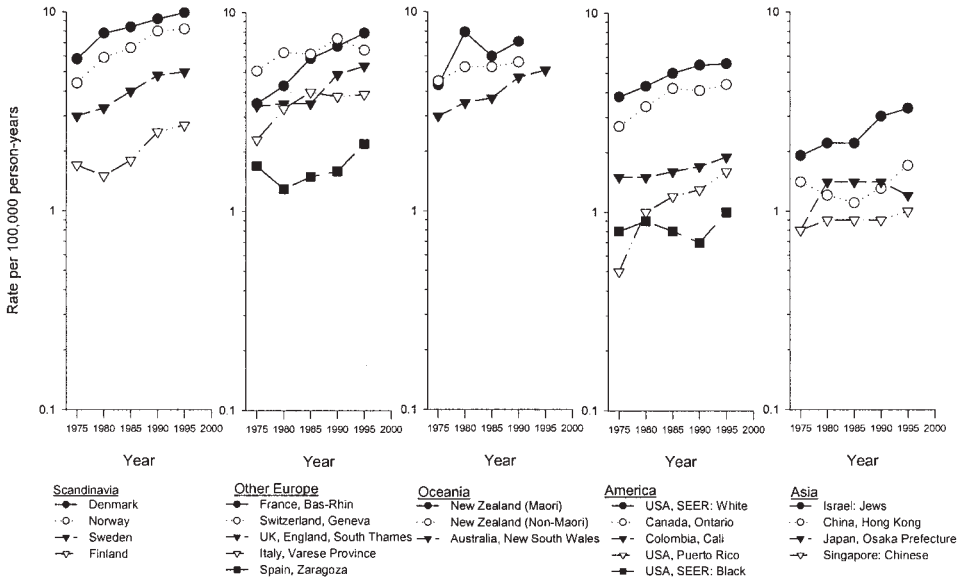

FIGURE 2 – Trends in age-standardized testis cancer rates by continent and area from time periods 1973–1977 to 1993–1997.

among males aged <30 years and 30–49 years rose in nearly all populations; among older males, rates increased among some populations (Nordic countries, Bas-Rhin, South Thames, New Zealand non-Maori) but not others. A plateau in rates among males aged <30 years was apparent for the populations of Ontario, Denmark, and Bas-Rhin. In contrast, testis cancer incidence in Finland rose considerably in the youngest age group and was nearly as high as that for males aged 30–49 during the time period 1993–1997. The rates for Osaka males aged <30 years and 50+ years rose until the 1983–1987 time period and declined thereafter.

Discussion

Testis cancer incidence among the 21 selected populations varied more than 10-fold; as expected, the highest rates were found in populations of European ancestry, whereas Asian, African, Latin American and U.S. black populations experienced low rates of disease. The proportional distribution of histologic subtypes did not differ systematically between high-incidence and low-incidence populations. During the period 1973–1997, the incidence increased among all populations examined, although the increases were generally strongest and most consistent among high-incidence populations.

The increase in testis cancer rates among initially low-incidence populations was generally less rapid compared to the rise among high-incidence populations. An exception is Puerto Rico, which experienced the most rapid increase between 1973–1977 and 1993–1997 (220%) among the selected populations. This increase is largely a function of the extremely low rate reported for the 1973–1977 time period, the lowest recorded among all populations, based on 34 cases. Cases and rates doubled from 1973–1977 to 1978–1982, with the increases subsequently more moderate and consistent. The number of cancers in Puerto Rico for 1973–1977

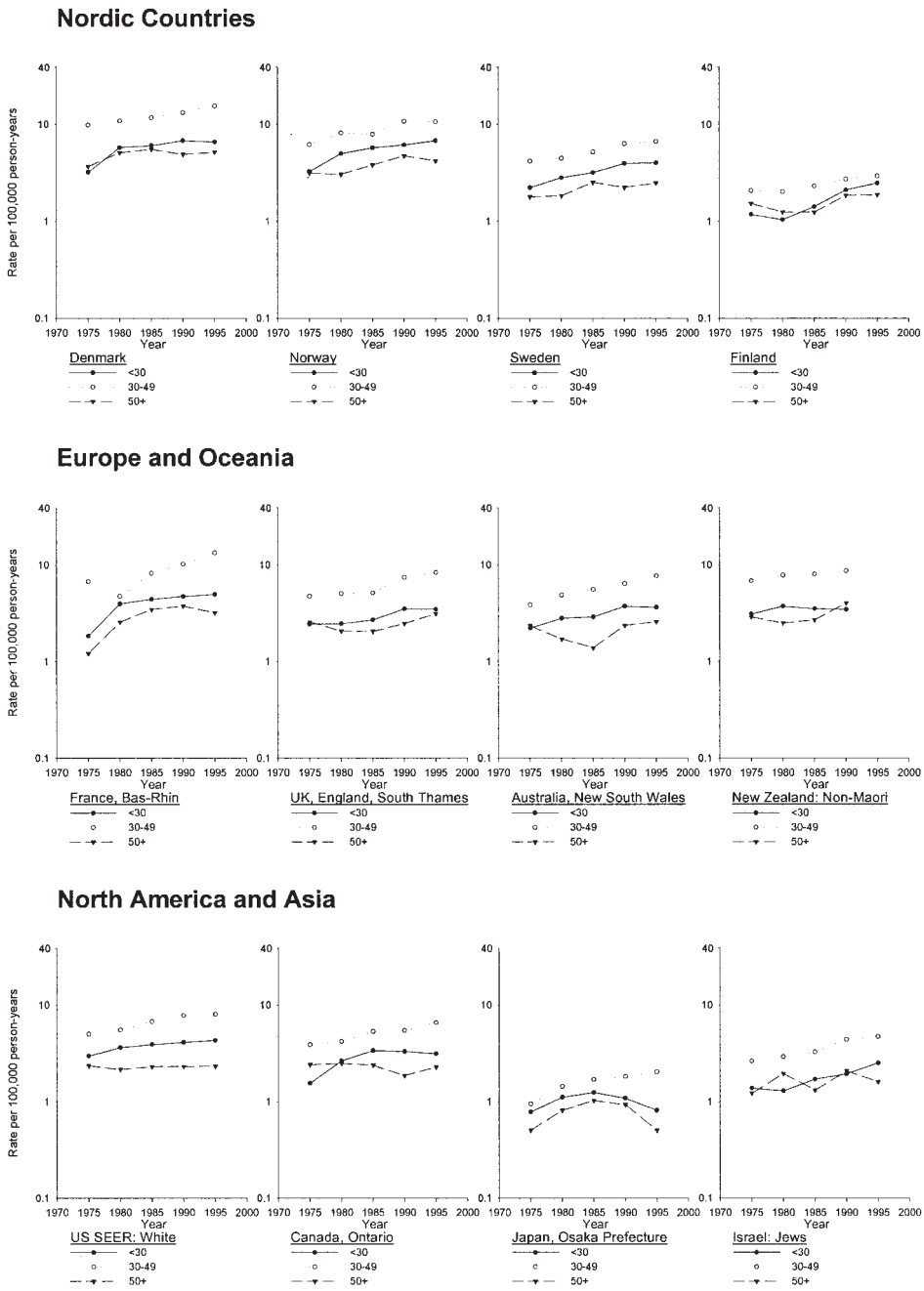

FIGURE 3 – Trends in age-standardized testis cancer rates by age group (<30, 30–49, 50+) for selected populations from time periods 1973–1977 to 1993–1997.

may have been underascertained; excluding that time period, the percent increase between 1978–1982 and 1993–1997 is equal to the average percent increase across all populations (60%).

One geographic region for which there is no clear evidence of an increase in incidence is Southeast Asia. Rates in Hong Kong decreased until 1983–1987 and increased thereafter, whereas rates among Singapore Chinese increased only slightly between 1973 and 1997. Testis cancer rates in Osaka among ages 15–64 previously were reported as having doubled between 1970 and 1987.[4] Our analysis indicates that rates increased only between 1973–1977 and 1978–1982 and stayed level thereafter. It is unclear what factors explain the observed Osaka trends. One possibility is that the observed initial increase in incidence is an artifact of improvements in completeness of ascertainment, as is suggested by the decrease in the percentage of testis cancer cases ascertained from a death certificate only (from 16% in 1973–1977 to 9% in 1978–1982 and around 2% or less thereafter).[2,9,14–16]

There have been reports suggesting that testis cancer rates in Denmark, U.S. whites and the Canton of Vaud, Swizerland, may have begun to plateau in recent years, possibly as a result of a stabilization of incidence rates among recent birth cohorts.[5,6,19] The trends for Denmark, U.S. whites, Ontario, Osaka and Varese do appear to be leveling off, although the changes involved are small (an exception is Osaka, discussed previously). The examination of trends in incidence among younger populations can be particularly informative in assessing the emergence of a change in secular trends.[20] Our analysis of trends by age group suggests that a flattening of testis cancer trends among younger males has occurred for some populations (notably Denmark, Ontario and Bas-Rhin), although no such plateau was apparent among U.S. whites. These data do not offer persuasive evidence of a change in the secular

trend of testis cancer; however, future trend analyses extended to the next 5-year period (1998–2002, expected to be available from IARC by 2007) may provide more insight into the question of whether testis cancer rates are stabilizing in some populations.

Unlike in other Nordic registries, testis cancer incidence among Finnish males aged less than 30 increased rapidly. This is consistent with the study by Bergstrom *et al*. that showed that the increase in testicular cancer incidence with successive birth cohorts born after 1945 is far stronger for Finland compared to other Nordic countries.[21] One possible explanation is the rapid improvement in living conditions that occurred in Finland after the Second World War and the corresponding improvement in childhood nutrition.[22] Interestingly, the narrowing in testis cancer rates among young men in Finland and Sweden parallels a recent narrowing between the 2 countries in body height.[22] Height is known to be associated with childhood nutrition[23] and has been suggested to be a risk factor for testicular cancer.[24]

There is a variety of factors that could account for the general increases in testis cancer incidence observed. One possibility is that changes in diagnostic patterns are responsible, with more intensive surveillance leading to increased detection of early, asymptomatic disease. This explanation is implausible, however, given the absence of screening programs targeting the general population, the steady increase in incidence across time periods and the fact that most testis cancers are symptomatic at diagnosis. Changes in coding practices can also result in artifactual changes in disease incidence. However, the ICD-9 classification is identical to the ICD-8 coding for testis cancer. As well, the continuously increasing trend observed for most populations is inconsistent with an effect of any coding change.

A third possibility is that there have been changes over time in the prevalence of important risk factors for testis cancer. Several studies have described secular increases in testis cancer incidence to be predominantly driven by a birth cohort effect.[6,8,21,25] Such a pattern is consistent with an increase in the prevalence of causal factors and/or a decrease in protective factors. However, it is unclear what risk factors may be responsible for the increase in incidence. High socioeconomic status has been reported to be associated with an increased risk of testis cancer.[26,27] Given the improvements in the standard of living for many populations over the past century, it is possible that some factors accompanying these changes in lifestyle is responsible for the increase in incidence. Increased maternal age and low parity have been suggested to be risk factors for testis cancer in some studies[28–30] and have also increased in prevalence in many populations over the past half-century.[31] These factors are known to be associated with the presence of elevated maternal estrogen levels during pregnancy, a condition hypothesized to increase the risk of abnormal gonadal development leading to testis cancer.[32,33] Analyses of ecologic data have supported the hypothesis that testis cancer trends may be due to changes over time in maternal smoking levels.[34,35] However, maternal smoking has not been linked to testis cancer risk in the majority of case-control studies conducted to date.[30,36–39] Low birth weight and low gestational age have been reported in some studies to increase testis cancer risk.[29,36,40] With the improvements in recent decades in the survival of premature infants,[41] an increased frequency of testis cancers arising among survivors of premature birth may have contributed to an increase in testis cancer rates. Reported decreases in age at puberty and early-life infections are also consistent with an increase in testis cancer rates, given the suspected etiologic relevance of male sex hormone levels and of viral infections in late childhood, respectively.[42,43] Lastly, there is speculation that ''endocrine modulators,'' exogenous compounds with weak estrogenic or antiandrogenic effects, may increase the risk of testis cancer by interfering with *in utero* gonadal development.[44] However, there is little epidemiologic evidence supporting this hypothesis.[44]

In summary, our analysis of published CI5 data suggests that testis cancer rates increased between 1973 and 1997 in most populations worldwide, although the increase is strongest and most consistent among high-incidence populations. There is also some indication that rates may be beginning to level off in some populations. The worldwide increase in testis cancer incidence may be due to changes in the prevalence of important risk factors; epidemiologic investigations describing between-population differences and within-population trends in the prevalence of suspected risk factors may yield important insight into the causes of the widespread increase in testis cancer risk.

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